nephritis, the condition has not been produced experimentally. However, a few very interesting observations have been made. In very many of the cases of extreme intoxication, as well as in those cases where excision of the kidney substance has been performed, there is marked and fairly early vomiting and diarrhoea. The metabolic studies of Pearce, Hill and Eisenbrey,[30] as well as those of Siegel,[31] have shown that though there may be a definite decrease in the nitrogenous excretion of the kidneys, there is no corresponding increase in the amount of nitrogen excreted in the feces, so that the gastro-intestinal disturbances cannot be due to a vicarious excretion of nitrogenous substances into the bowel. Moreover, it has been shown that in certain cases of tubular nephritis, to which group uranium and chromate nephritis belong, there may be marked gastro-intestinal disturbance when there is no appreciable decrease in the nitrogenous excretion by the kidneys. From this it would seem that the gastro-intestinal disturbance must be due to some non-nitrogenous substance which is normally excreted by the tubular epithelium of the kidneys. In this connection Pearce [32] has shown that in the urine of normal dogs there is some substance which, when injected intravenously into normal dogs, will lower the blood pressure, that in the urine of dogs suffering from tubular nephritis this substance is absent, and that in the serum of dogs suffering from tubular nephritis this substance is present. He has not yet determined what this depressor substance is, but it is possible that its vicarious excretion into the bowel is responsible for the gastro-intestinal disturbances, and it is also possible that it is this or some similar substance which has to do with the onset of uremic symptoms.

In this necessarily very brief resumé of the subject, I have been unable to do more than barely touch upon some of the more interesting and recent observations that have been made. However, I hope I have been able to show that by means of experimental research, observations are being made which must eventually do much towards clearing up many of the unexplained phenomena of Bright's Disease.

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NEPHRITIS OF BACTERIAL ORIGIN.

By JAMES J. HOGAN, M. D., Vallejo, Cal.

The presence of microorganisms in the urine of a person suffering with all the clinical symptoms of nephritis does not prove that they are the direct cause of the attack, but it is reasonable to suppose that either the organism itself or the toxins it produces are causal factors in the production of the types of nephritis that clear rapidly on the disappearance of the organism.

Certain types of microorganisms are capable of producing toxic substances to a varying extent; for instance, the different staphylococci belong to the type of the least toxic, while some types of the streptococci, pneumococci and others, although present in smaller numbers, produce toxins of the most virulent type. This would explain why the presence of some in the urine produce no symptoms and others give a true picture of nephritis.

It has been proven by Richter, Heinecke, Dickson, Pearce and others that any type of nephritis, from the most acute to the chronic interstitial form, can be produced by the injection of varying doses

of some salts, as uranium nitrate, arsenic and potash chromate, and the acuteness or chronicity of the condition could be regulated by the size of the dose, the interval, and the period over which it was continued. That this same thing does occur in the course of many of the acute infections, producing acute nephritis, is not disputed by many, and that the continuation of the infection may produce any of the chronic types is reasonable to assume.

Now the point I wish to make is this: nephritis from the most acute type to that of chronic character is simply a symptom of a circulatory change taking place in the kidneys, and the intensity of the attack is in proportion to the amount or degree of virulence of the toxin.

Martin H. Fisher's work on the "Nature and Cause of Edema" gives us the most reasonable explanation of the train of symptoms that follow the injection of the salts of some metals and the circulation of the toxic products of bacterial growth. He has shown that the first effect is the production of an acidosis in the part, this producing a change in the colloids; they absorb more water and swell, the consequent result is a mechanical process. The kidney being an organ surrounded by a firm capsule, this swelling can only go so far when the pressure of the increased fluid forces the blood out of the kidney and a partial or complete anuria results. As the blood is unable to get rid of its toxic contents through the kidneys, they circulate through the system and local and general edema result from the same cause as started the original trouble in the kidneys.

I mention Fisher's work to you for the reason that he has proven to the satisfaction of the most prominent physiologists in Europe that his explanation of the nature and cause of edema is the right one, and also to call your attention to the fact that his work has never been mentioned in the recent work of men who have been producing nephritis and edema in the experimental laboratory.

In the past two and a half years I have made a bacteriological examination of 1225 specimens of urine, and of this number 26 were from different types of nephritis. Of the 26 we demonstrated by culture, organisms of different types in 17 and in 9 no growth was obtained. The organisms found were the colon type, pneumococcus, streptococcus type, staphylococcus, typhoid and a diplococcus that I have not been able to classify. This last one I found in three cases, one of hematuria and two chronic types with symptoms of the interstitial form. It grows freely on all media, is gram positive, surface growth on agar resembling the colon type and decolorizes and peptonizes litmus milk. Nicholls, in the Montreal Medical Journal, 1899, p. 161, states that in 45 cases of chronic nephritis he found a minute diplococcus in 29.

That my findings have not been out of line with other workers in the same field is shown by identical findings of Nicholls, the co-editor with Adami in his work on Systemic Pathology which was published recently. He found in 32 cases of acute nephritis, bacteria present in 28, and in 45 cases of chronic nephritis of all types, a diplococcus in 29 and a bacillus in 4. The source of the bacteria in the chronic types he says is difficult to determine, but he remarks that in 41 per cent of these cases there was a definite history of preceding gastro-enteric disturbance.

Treatment. Of the treatment of the acute types occurring in the course of the acute infectious diseases there is little to say. As a rule they clear without leaving any permanent change. But in those of the more chronic types we should make it a rule to culture the urine, and in the event of obtaining a growth, make an autogenous vaccine. While the results of vaccine treatment are not always brilliant, the perfectly marvelous clearing up of all symptoms in some cases that have resisted all other lines of treatment should lead us to give this type of patient at least a chance.

In closing I wish to say that culturings of catheter specimens of urine in patients suffering with nephritis and the subsequent use of an autogenous vaccine, if microorganisms are found present, will frequently give you the most brilliant results in a type of case that formerly was hopeless.

HEMATURIA; AN INITIAL SYMPTOM OF CHRONIC NEPHRITIS.*

By R. L. RIGDON, M. D., San Francisco.

Under the headings of essential hematuria, idiopathic hematuria, symptomless hematuria, etc., have been grouped a class of renal bleeding, the cause or causes of which were unknown. By the aid of newer methods of examination, chiefly ureteral catheterization, and more careful and complete microscopic examination of extirpated or autopsied kidneys, much additional light has been shed upon this obscure field of renal pathology, and to-day we are able to separate some of these so-called idiopathic hematurias into rather definite groups and to more properly classify them.

Much has been done also tending to show the exact origin of the hemorrhage in these cases. It has been proven that the hemorrhage may occur (1) from the substance of the kidney, (2) from a renal papillæ, or (3) from the mucous membrane of the kidney pelvis.

Our paper to-day has to deal with hematuria of interstitial nephritis. We have further limited our paper to renal hematuria as an initial symptom of chronic nephritis, thus excluding those hemorrhages that occur late in the course of the disease and are so well known. That the initial symptom of interstitial nephritis might be a hematuria has been known in a vague way for many years but not until 1897 was the definite causal relationship somewhat fully worked out and the claim established. Since that time so many cases have been examined and carefully reported that to-day the question is raised out of the domain of argument and placed among those that are admittedly established.

Another question which gave occasion for much argument was as to whether chronic interstitial nephritis was always a bilateral affair. At first the affirmative was almost universally upheld, but after the introduction of the cystoscope, and the great

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